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Is there a threshold intensity for aerobic training in cardiac patients?

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ABSTRACT

SWAIN, D. P., and B. A. FRANKLIN. Is there a threshold intensity for aerobic training in cardiac patients? *Med. Sci. Sports Exerc.*, Vol. 34, No. 7, pp. 1071–1075, 2002. **Purpose:** Recent guidelines have recommended the use of a percentage of oxygen uptake reserve ($\dot{V}O_2R$) for prescribing aerobic exercise intensity for cardiac patients. Moreover, these guidelines suggest that a threshold intensity may exist, below which no improvement in peak oxygen uptake ($\dot{V}O_{2peak}$) occurs. The purpose, therefore, was to translate the intensity of aerobic exercise in previous training studies using cardiac patients into $\%\dot{V}O_2R$ units, and determine whether a threshold intensity exists. **Methods:** Twenty-three studies, using 28 groups of aerobically trained cardiac patients, were identified in which $\dot{V}O_{2peak}$ was measured before and after training by gas exchange. Intensity of exercise was variously described as a percentage of $\dot{V}O_{2peak}$, percentage of peak heart rate (HR_{peak}), percentage of heart rate reserve (HRR), or percentage of peak workload. These intensities were translated into equivalent units of $\%\dot{V}O_2R$. **Results:** Of the 28 groups of patients, three failed to show significant improvements in $\dot{V}O_{2peak}$. These groups exercised at intensities corresponding to 47–55% of $\dot{V}O_2R$. However, six other groups exercised at comparable intensities (i.e., 42% to 55% of $\dot{V}O_2R$) and experienced significant increases in $\dot{V}O_{2peak}$. Other confounding variables in these studies were similar, including the initial $\dot{V}O_{2peak}$ of the subjects, suggesting that the failure of three groups to significantly improve aerobic capacity was due to their small sample size. **Conclusion:** No threshold intensity for aerobic training was identified in cardiac patients, with the lowest intensity studied being approximately 45% of $\dot{V}O_2R$. It is possible that intensities below this value may be an effective training stimulus, especially in extremely deconditioned subjects, but further research is needed to test that possibility and to determine whether a threshold exists. **Key Words:** EXERCISE, CARDIAC REHABILITATION, MAXIMUM OXYGEN CONSUMPTION

According to recent guidelines from the American College of Sports Medicine (ACSM), cardiac patients should exercise above a minimum intensity to achieve a cardiorespiratory training effect, i.e., a "threshold" intensity (10). Furthermore, these guidelines suggest that the threshold for cardiac patients is most likely between 40% and 50% of oxygen uptake reserve ($\dot{V}O_2R$). However, the value for this threshold is based upon the ACSM's 1998 position stand that reviewed studies performed on healthy subjects (24). Thus, there is a need to determine whether a threshold training intensity exists in cardiac patients and, if so, its value.

$\dot{V}O_2$ reserve is the difference between resting and maximal oxygen uptake. Studies in healthy individuals (30,31) and in cardiac patients (3) have shown that percentages of heart rate reserve (HRR) more accurately reflect $\%\dot{V}O_2R$ than $\%\dot{V}O_{2max}$. Consequently, $\%\dot{V}O_2R$ is now preferred over $\%\dot{V}O_{2max}$ for prescribing exercise intensities that are based on oxygen uptake (10,24).

Recently, the authors analyzed previous training studies of healthy subjects to determine whether a threshold intensity exists in that population, and identified a threshold at 45% $\dot{V}O_2R$ for individuals with initial aerobic capacities of at least 40 $mL\cdot min^{-1}\cdot kg^{-1}$, but no threshold for individuals with lower capacities, although the lowest training intensities examined were approximately 30% $\dot{V}O_2R$ (29). Given that the threshold in healthy subjects with low fitness levels, if one exists, is less than 30% $\dot{V}O_2R$, it seems likely that any threshold in the typically deconditioned cardiac population would be no more than this value. The purpose of the current study was to analyze previous training studies of cardiac patients, to translate the reported intensity of training into $\%\dot{V}O_2R$ units, and to determine whether there is a threshold for improvement in aerobic capacity.

METHODS

Studies that evaluated the effect of aerobic training on the peak $\dot{V}O_2$ of cardiac patients were identified from a comprehensive 1995 review of exercise-based cardiac rehabilitation performed by the U.S. Department of Health and Human Services (34), from a MEDLINE search for studies published thereafter, and by checking the reference lists of all reports obtained in the first two steps. There were several cases in which two or more investigations identified in the initial search used a single data set, publishing different aspects of the results of training in separate papers. In these cases, only one study was cited. Also, there were instances where a study was published and later added more subjects in a subsequent publication. In such cases, only the most recent, i.e., most complete, study was cited.

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TABLE 1. Aerobic training studies ($N = 23$) in cardiac patients.

Study	N	Age (yr)	Sex	Initial $\dot{V}O_{2peak}$ (*)	Mode	Freq. (N/wk)	Duration (min)	Length (wk)
Kasch and Boyer (17)	11	32–63	M	19.9	Bike	4	53	26
Costill et al. (6)	24	avg 52	M	20.0	TM	4	30	12
Franklin et al. (12)	16	45–59	M	23.5	Walk-jog	3	15–30	12
Ehsani et al. (9)	8	42–62	M	26	Walk-jog, bike	3–5	30–60	52
Dressendorfer et al. (7)	8	37–64	M	29.4	Walk	3	40	14–20
Vanhees et al. (32)	15	avg 49	M, with β	22.3	Various	3	50	13
	15	avg 51	M, no β	21.7				
Roman et al. (25)	93	32–71	87M, 6F	1.2 L·min^{-1}	Various	3	30	26
Myers et al. (21)	48	35–65	M	25.3	Various	3	45	52
Froelicher et al. (13)	59	35–65	M	26	Arm, leg devices	?	45	52
Sullivan et al. (27)	19	avg 53	not stated	26.2	Various	2.2	38	52
Ehsani et al. (8)	25	31–69	24M, 1F	23	Walk-jog, bike	3–5	40–60	52
Blumenthal et al. (2)	23	34–63	M	23.4	Walk-jog	3	30–45	12
	23	28–66		21.8				
Sullivan et al. (28)	12	avg 54	not stated	16.8	Various	4	60	20
Oldridge et al. (23)	12	avg 52	M	22.5	Bike, walk	2	53	12
Haennel et al. (14)	8	avg 52	M	21.1	Cycle	3	24	8
Meyer et al. (20)	12	45–75	M	13.0	Cycle	5	22	6
Coats et al. (5)	17	47–74	M	13.2	Bike	5	20	8
Keyser et al. (19)	15	avg 65	13M, 2F	15.6	Arm/leg cycling	3	30	12
Oberman et al. (22)	103	30–69	M	25.3	Various	3	60	52
	83			24.2				
Adachi et al. (1)	10	26–62	9M, 1F	21.9	Walk	5	30	8
	11	48–72	10M, 1F	18.7				
Jensen et al. (16)	93	30–67	M	25.3	Walk-jog, cycle	3	45	52
	93			24.3				
Keteyian et al. (18)	15	avg 52	M	16.0	Various	3	33	24
Stewart et al. (26)	11	avg 57	M	21.2	Arm/leg cycling	3	23	10

Only studies that measured pre- and post-training $\dot{V}O_{2peak}$ by using gas exchange techniques were included. This eliminated numerous studies that evaluated the effects of training on the estimated functional capacity of cardiac patients. For the purposes of this research, cardiac patients were those diagnosed with coronary artery disease, in most cases having experienced a myocardial infarction, but also including those having undergone coronary revascularization (14), those with left ventricular dysfunction (heart failure) (5,18,20,28), and those demonstrating significant ($\geq$1.5 mm), exercise-induced, ST-segment depression (6). The greatest $\dot{V}O_2$ attained during exercise testing is referred to in this analysis as $\dot{V}O_{2peak}$ rather than $\dot{V}O_{2max}$ because true maximal values are often not attained during symptom-limited tests of cardiac patients.

Exercise intensities in the training studies were originally reported as percentages of $\dot{V}O_{2peak}$, percentages of peak heart rate (HR_{peak}), percentages of HRR, or percentages of peak workload. These measures of exercise intensity were converted to $\%\dot{V}O_2R$ units by the methods described in a related study on healthy subjects (29) and are summarized below.

In those studies reporting training intensity as $\%\dot{V}O_{2peak}$ (6–9,13,16,22,28,32), this percentage was multiplied by the reported mean initial $\dot{V}O_{2peak}$ to yield the gross exercise $\dot{V}O_2$ in mL·min^{-1}·kg^{-1}. Then, $\%\dot{V}O_2R$ was calculated from the following formula: $\%\dot{V}O_2R$ = (gross exercise $\dot{V}O_2$ -3.5)/($\dot{V}O_{2peak}$ -3.5), where 3.5 mL·min^{-1}·kg^{-1} was assumed to be the average resting $\dot{V}O_2$ of the subjects.

In those studies reporting training intensity as $\%HR_{peak}$ (1,5,12,17,23,25,26), this was converted to $\%\dot{V}O_2R$ using the formula: $\%\dot{V}O_2R = 1.667(\%HR_{peak}) - 70\%$. As described previously (29), this formula was derived independently from two different $HR/\dot{V}O_2$ data sets in healthy adults. The two data sets yielded nearly identical formulas, and the aerobic capacity of the subjects had only a minor effect on the relationship. Because patients with heart disease exhibit the same highly linear relationship between HR and $\dot{V}O_2$ across the range of rest to maximum exercise as do healthy subjects, even in the presence of beta-blockers and/or calcium antagonists (3,4,15), this formula should provide a reasonable estimate of $\%\dot{V}O_2R$ in the cardiac population.

For studies reporting training intensity as %HRR (2,14,18,19,21,27), these were assumed to provide equivalent values in $\%\dot{V}O_2R$ units, as previously shown for healthy adults (30,31) and cardiac patients (3). Similarly, one study reported the training intensity as a percentage of peak workload (20), and this value was assumed to be equivalent to $\%\dot{V}O_2R$.

RESULTS

Table 1 presents a summary of the 23 studies that were analyzed. All but seven of the studies utilized a sedentary control group. One of the studies compared a group of subjects on beta-blockade with a group not on beta-blockade, and reported similar responses to training (32). Four other studies, described below, compared groups of subjects

TABLE 1—*Continued*

Reported Intensity	%$\dot{V}O_2R$	Increase in $\dot{V}O_{2peak}$	Test Mode and Termination Criteria	Other Comments
69–80% HR_{peak}	45–63%	39% at 3 months, 54% overall	Bike; none stated	No control group; intensity increased from 69% to 80% HR_{peak} at 3 months
68% $\dot{V}O_{2peak}$	61%	21%	TM; symptoms or 1.5 mm ST depression	
73% HR_{peak}	51%	13%	Bike; fatigue or symptoms	No control group
50–95% $\dot{V}O_{2peak}$	42–94%	42%	TM; plateau for 6 subjects, symptom-limited for 2	Intensity increased from 50–60% to 70–95% $\dot{V}O_{2peak}$ at 3 months, but peak not reported at that point
60% $\dot{V}O_{2peak}$	55%	(3%) ns	TM; fatigue (mean RERmax was 1.08)	No control group
70% $\dot{V}O_{2peak}$	64%	36%	Bike; fatigue or symptoms (mean RERmax was 1.06)	No sedentary control group
		34%		
70% HR_{peak}	47%	50%	Bike; fatigue or symptoms	
60% HRR	60%	6%	TM; fatigue or symptoms	
77% $\dot{V}O_{2peak}$	73%	9%	TM; fatigue or symptoms	Intensity was highly variable, 77% is mean
64% HRR	64%	1%	TM; fatigue or symptoms (mean RERmax was 1.12)	Increased $\dot{V}O_{2peak}$ was sig. relative to controls, who declined 7%
60–90% $\dot{V}O_{2peak}$	53–88%	37%	TM; plateau for 15 subjects, symptom-limited for 10	Intensity increased from 60–70% to 70–90% $\dot{V}O_{2peak}$ at 3 months, but peak not reported at that point
70% HRR	70%	13%	TM; fatigue or symptoms	Increase not diff. between groups; total work not equated
<45% HRR	<45%	15%		
75% $\dot{V}O_{2peak}$	68%	23%	Bike; fatigue or symptoms (mean RERmax was 1.32)	No control group, all subjects with CHF
73% HR_{peak}	52%	18%	Bike; fatigue or symptoms	
70% HRR	70%	20%	Bike; plateau or 90% pred. HRmax or symptoms	All patients with CABG
75% W_{peak}	75%	12%	Bike; fatigue	Crossover control, all subjects with CHF
70% HR_{peak}	47%	18%	Bike; fatigue	Crossover control, all subjects with CHF
78% HRR	78%	9%	Bike; fatigue	No control group
85% $\dot{V}O_{2peak}$	82%	10%	TM; fatigue or symptoms	Sig. diff. between groups, but total work not equated
50% $\dot{V}O_{2peak}$	42%	9%		
81% HR_{peak}	65%	17%	Bike; fatigue or symptoms	Total work not equated
70% HR_{peak}	47%	(9%) ns		
85% $\dot{V}O_{2peak}$		13%	TM; age-predicted HRmax, symptoms	Sig. diff. between groups, but total work not equated
50% VO_{2peak}		9%		
60–80% HRR	60–80%	14% at 12 wk, 16% overall	Bike; fatigue or symptoms (RERmax was 1.13)	All subjects with CHF; intensity increased from 60% to 80% HRR after 2 wks in some subjects
75% HR_{peak}	55%	(8%) NS	Bike; RPE of 19–20 or RER $>$ 1.1	No control group

* $mL \cdot min^{-1} \cdot kg^{-1}$; TM, treadmill; RER, respiratory exchange ratio; M, male; F, female; β, beta-blocker therapy; CHF, congestive heart failure; CABG, coronary artery bypass graft surgery; RPE, rating of perceived exertion; W, workload.

at two different intensities. Thus, a total of 28 subject groups were evaluated. The range of durations, frequencies, and length of programs (i.e., in weeks) was too diverse to allow an analysis of these factors.

As seen in Table 1, three of the training groups failed to demonstrate statistically significant increases in $\dot{V}O_{2peak}$, although they exhibited numerically greater values after training (by 3%, 8%, and 9%) and had small numbers of subjects (8, 11, and 11, respectively). These three studies utilized exercise intensities ranging from 47% to 55% of $\dot{V}O_2R$. Six other studies that utilized similar training intensities (from 42% to 55% of $\dot{V}O_2R$) reported significant increases in $\dot{V}O_{2peak}$. The initial mean $\dot{V}O_{2peak}$ values of the three groups that did not show improvement were 18.7, 21.2, and 29.4 $mL \cdot min^{-1} \cdot kg^{-1}$, which overlapped the six groups that did show improvement (13.2, 21.8, 22.5, 23.5, and 24.2 $mL \cdot min^{-1} \cdot kg^{-1}$, and one group with 1.2 $L \cdot min^{-1}$, body mass not provided). Thus, the failure of three groups to demonstrate statistically significant improvements is most likely due to their small sample size, rather than being an indication of a threshold intensity.

The mode of exercise and the duration (minutes per session), frequency (sessions per week), and length (number of weeks) of training varied greatly among the 23 studies, precluding any conclusions regarding the influence of these variables. Four studies each utilized two groups of different training intensities, making it possible to compare the effects of intensity on improvements in aerobic capacity. Three of these studies reported a significant difference between groups for the increase in $\dot{V}O_{2peak}$, and in each case the higher intensity group exhibited the greater relative improvement. However, in each of these studies the two groups used the same exercise duration; consequently, the higher intensity group performed a greater total amount of training.

DISCUSSION

This analysis found no evidence of a threshold intensity for aerobic training of cardiac patients, i.e., there was not an intensity that could be identified as a minimum intensity for eliciting improvement in peak oxygen uptake. Because the lowest intensities used in any of the studies approximated 45% of $\dot{V}O_2R$, it is possible that intensities below this value are capable of improving $\dot{V}O_{2peak}$. However, it is also possible that a threshold exists at or below 45% of $\dot{V}O_2R$. Consequently, until further research is conducted using lower training intensities, 45% of $\dot{V}O_2R$ should be considered the minimal effective intensity for eliciting improvements in cardiorespiratory fitness in patients with coronary heart disease.

Most of the studies included in this analysis had little or no criteria for the attainment of $\dot{V}O_{2max}$. Generally, incremental exercise tests were stopped due to volitional fatigue or when patients demonstrated adverse signs or symptoms. Their results were included in the studies' data despite the

fact that criteria for $\dot{V}O_{2max}$, such as a plateau in $\dot{V}O_2$ or the attainment of a respiratory exchange ratio (RER) of 1.10 or more, were not achieved. This commonly occurs when exercise testing cardiac patients and is why their highest achieved $\dot{V}O_2$ is often referred to as $\dot{V}O_{2peak}$ in contradistinction to $\dot{V}O_{2max}$. This has two consequences to the current analysis: interpreting the increase in aerobic capacity following training, and expressing the exercise intensity during training as a percentage of maximum capacity.

True $\dot{V}O_{2max}$ was often not attained during testing, as indicated in some studies by significant posttraining increases in peak RER or peak HR. Thus, the increase in aerobic capacity after training could be due in part to greater effort by the patients, a reduction in their symptoms, or both, as opposed to an increase in aerobic capacity *per se*. Keteyian et al. (18) specifically addressed this issue, and attributed 46% of the posttraining increase in $\dot{V}O_{2peak}$ in their study to the increase in HR_{peak}. On the other hand, it seems reasonable to conclude that some physiological increase in aerobic capacity does occur with training in cardiac patients (e.g., 54% of the increase in $\dot{V}O_{2peak}$ in Keteyian et al.'s study). Some of the studies in this analysis had strong physiological indicators of maximal effort. In Ehsani et al.'s 1986 study (8), 15 of 25 subjects achieved a plateau in oxygen uptake. By using an unconventionally high exercise intensity during training (up to ~90% of $\dot{V}O_2R$), a 39% increase in true $\dot{V}O_{2max}$ was reported among the 15 subjects who exhibited plateaus. Furthermore, Ehsani et al. reported significant improvements in left ventricular function, such as a greater ejection fraction during maximal exercise. Four studies reported mean maximal RERs above 1.10, and three of these obtained significant increases in aerobic capacity (18,27,28), whereas the fourth reported a trend ($P = 0.15$) for an 8% increase (26). Therefore, although not all of the increase in $\dot{V}O_{2peak}$ in the studies included in this analysis can be attributed to a physiological increase in aerobic power, properly performed aerobic training clearly increases true $\dot{V}O_{2max}$ in cardiac patients.

Because a true maximal effort was probably not attained in many of the studies, the reported exercise intensities are likely overestimates of the actual ranges, e.g., if a study reported an exercise intensity as 70% of HR_{peak}, this value is likely higher than the corresponding percentage of HR_{max}. The translated values in $\%\dot{V}O_2R$ units are also likely to be overestimated. Consequently, the minimal effective intensity identified in this analysis, 45% of $\dot{V}O_2R$, must be considered in light of the fact that maximal $\dot{V}O_2$ was not known. If a true $\dot{V}O_{2max}$ were known, then this value would probably be much lower.

In a recent analysis of training studies performed with healthy adults, the authors found that 45% of $\dot{V}O_2R$ was a threshold intensity for individuals with initial $\dot{V}O_{2max}$ values of at least 40 $mL \cdot min^{-1} \cdot kg^{-1}$, whereas 30% of $\dot{V}O_2R$ was the minimal effective intensity for those with lower initial capacities (29). The lower fit subjects did not demonstrate a threshold *per se*, as no studies used intensities below 30% of $\dot{V}O_2R$. The current analysis in cardiac patients, with initial $\dot{V}O_{2peak}$ values ranging from 13.0 to 29.4 $mL \cdot min^{-1} \cdot kg^{-1}$, found no threshold intensity, consistent with the finding in healthy subjects with low initial fitness, although the lowest intensities evaluated in cardiac patients approximated only 45% of $\dot{V}O_2R$.

Finally, we recognize that training effects in the present analysis were narrowly defined to signify improved cardiorespiratory fitness, rather than global health outcomes. Accordingly, substantial health benefits may still be achieved at exercise levels that are below the minimal effective training intensity identified here (i.e., $< 45\%$ $\dot{V}O_2R$), provided that the frequency and duration of training are appropriate. Research has shown that numerous health benefits can be derived at more moderate exercise intensities, that is, at intensities below those commonly prescribed for cardiorespiratory conditioning. These include favorable changes in bone density, glucose tolerance, and coronary risk factors, as well as a reduction in cardiovascular-related mortality (11). There are also intriguing data to suggest that small, insignificant group increases in aerobic fitness (i.e., 3 to 9%) may, on an individual basis, be associated with meaningful reductions in subsequent coronary events, especially in cardiac patients with low baseline $\dot{V}O_{2peak}$ (33).

CONCLUSION

Our analysis suggests that 45% of $\dot{V}O_2R$ should currently be considered the minimal effective intensity for improving aerobic capacity in cardiac patients. In studies using cardiac patients, $\dot{V}O_2$ reserve is generally the difference between resting and peak $\dot{V}O_2$ rather than resting and maximal $\dot{V}O_2$. Thus, 45% of $\dot{V}O_2R$ may overestimate the minimal effective intensity if true $\dot{V}O_{2max}$ is known. Studies that used higher versus lower intensities of training generally obtained greater improvements in $\dot{V}O_{2peak}$ with the higher intensities; however, the total amount of work was not equated between groups. Future research with some groups exercising at less than 45% of $\dot{V}O_2R$, with total work equated between groups, and with criteria established for the attainment of maximal effort for pre and post testing, should help to clarify these issues.

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